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=> file medline hcaplus biosis biotechds embase
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

FILE 'MEDLINE' ENTERED AT 17:51:52 ON 23 JUN 2006

FILE 'HCAPLUS' ENTERED AT 17:51:52 ON 23 JUN 2006
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FILE 'BIOTECHDS' ENTERED AT 17:51:52 ON 23 JUN 2006
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FILE 'EMBASE' ENTERED AT 17:51:52 ON 23 JUN 2006
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=> s deaD gene disruption and carotenoid biosynthesis
L1            0 DEAD GENE DISRUPTION AND CAROTENOID BIOSYNTHESIS

=> s deaD gene and carotenoid biosynthesis
L2            0 DEAD GENE AND CAROTENOID BIOSYNTHESIS

=> s deaD gene and isoprenoid biosynthesis
L3            0 DEAD GENE AND ISOPRENOID BIOSYNTHESIS

=> s deaD gene and isoprenoid enzymatic biosynthesis
L4            0 DEAD GENE AND ISOPRENOID ENZYMATIC BIOSYNTHESIS

=> s deaD gene and isoprenoid
L5            0 DEAD GENE AND ISOPRENOID

=> s deaD gene
L6           41 DEAD GENE

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7            19 DUP REM L6 (22 DUPLICATES REMOVED)

=> s l7 and (disrupt? or delete)
L8             3 L7 AND (DISRUPT? OR DELETE)

=> d l8 1-3 ibib ab

L8   ANSWER 1 OF 3     MEDLINE on STN
ACCESSION NUMBER:    93264080     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 8494644
TITLE:               Defective glia in the Drosophila brain degeneration mutant
                     drop-dead.
AUTHOR:              Buchanan R L; Benzer S
CORPORATE SOURCE:    California Institute of Technology, Pasadena, California
                     91125.
CONTRACT NUMBER:     5F32NS0881-02 (NINDS)
                     EY09278 (NEI)
                     GM 40499 (NIGMS)
SOURCE:              Neuron, (1993 May) Vol. 10, No. 5, pp. 839-50.
                     Journal code: 8809320. ISSN: 0896-6273.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
```

ENTRY MONTH: 199306
ENTRY DATE: Entered STN: 2 Jul 1993
Last Updated on STN: 29 Jan 1996
Entered Medline: 18 Jun 1993

AB To understand better the cellular basis of late-onset neuronal degeneration, we have examined the brain of the drop-dead mutant of Drosophila. This mutant carries an X-chromosomal recessive mutation that causes severe behavioral defects and brain degeneration, manifested a few days after emergence of the adult. Analysis of genetically mosaic flies has indicated that the focus of the drop-dead mutant phenotype is in the brain and that the gene product is non-cell autonomous. We examined the adult drop-dead mutant brain prior to onset of symptoms and found that many glial cells have stunted processes, whereas neuronal morphology is essentially normal. Adult mutant glial cells resemble immature glia found at an earlier stage of normal brain development. These observations suggest that defective glia in the drop-dead brain may **disrupt** adult nervous system function, contributing to progressive brain degeneration and death. The normal drop-**dead gene** product may prevent brain degeneration by providing a necessary glial function.

L8 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:256307 HCAPLUS
DOCUMENT NUMBER: 136:293612
TITLE: Sequence of **deaD gene** from corynebacteria and use thereof in synthesis of L-lysine
INVENTOR(S): Farwick, Mike; Huthmacher, Klaus; Brehme, Jennifer; Pfefferle, Walter
PATENT ASSIGNEE(S): Degussa A.-G., Germany
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2002026787	A1	20020404	WO 2001-EP10772		20010918
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
DE 10047865	A1	20020418	DE 2000-10047865		20000927
AU 2001093821	A5	20020408	AU 2001-93821		20010918
EP 1320544	A1	20030625	EP 2001-974264		20010918
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
US 2002115161	A1	20020822	US 2001-963790		20010927
PRIORITY APPLN. INFO.:			DE 2000-10047865	A	20000927
			WO 2001-EP10772	W	20010918

AB The **deaD gene** of Corynebacterium glutamicum ATCC13032 encoding a DNA/RNA helicase is cloned for use in increasing the efficiency of fermn. of L-lysine by coryneform bacteria. The expression vector contg. **deaD gene** is constructed. Methods and culture media for fermentative prepn. of L-lysine with recombinant bacterial strains transformed with these vectors are also provided. **Disruption** of the **deaD gene** by integration mutagenesis using deaD expression vector increased the yield of lysine in

a Corynebacterium host from 13.57 g lysine/L at 7.6 OD660 to 16.31 g lysine/L at 12.2 OD660. The fermentatively prepd. L-lysine are useful in pharmaceutical industry and foodstuff industry and very particularly in animal nutrition.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 3 OF 3 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1999:263505 BIOSIS
DOCUMENT NUMBER: PREV199900263505
TITLE: Identification of virulence genes of Helicobacter pylori by random insertion mutagenesis.
AUTHOR(S): Bijlsma, J. J. E.; Vandenbroucke-Grauls, C. M. J. E.; Phadnis, S. H.; Kusters, J. G. [Reprint author]
CORPORATE SOURCE: Department of Medical Microbiology, Faculty of Medicine, Vrije Universiteit Amsterdam, Van der Boechorststraat 7, 1081, BT Amsterdam, Netherlands
SOURCE: Infection and Immunity, (May, 1999) Vol. 67, No. 5, pp. 2433-2440. print.
CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Jul 1999
Last Updated on STN: 15 Jul 1999

AB The complete genome of the gram-negative bacterial pathogen Helicobacter pylori, an important etiological agent of gastroduodenal disease in humans, has recently been published. This sequence revealed that the putative products of roughly one-third of the open reading frames (ORFs) have no significant homology to any known proteins. To be able to analyze the functions of all ORFs, we constructed an integration plasmid for H. pylori and used it to generate a random mutant library in this organism. This integration plasmid, designated pBCalpha3, integrated randomly into the chromosome of H. pylori. To test the capacity of this library to identify virulence genes, subsets of this library were screened for urease-negative mutants and for nonmotile mutants. Three urease-negative mutants in a subset of 1,251 mutants (0.25%) and 5 nonmotile mutants in a subset of 180 mutants (2.7%) were identified. Analysis of the **disrupted** ORFs in the urease-negative mutants revealed that two had **disruptions** of genes of the urease locus, ureB and ureI, and the third had a **disruption** of a unrelated gene; a homologue of deaD, which encodes an RNA helicase. Analysis of the **disrupted** ORFs in the nonmotile mutants revealed one ORF encoding a homologue of the paralyzed flagellar protein, previously shown to be involved in motility in Campylobacter jejuni. The other four ORFs have not been implicated in motility before. Based on these data, we concluded that we have generated a random insertion library in H. pylori that allows for the functional identification of genes in H. pylori.

=> d his

(FILE 'HOME' ENTERED AT 17:51:22 ON 23 JUN 2006)

FILE 'MEDLINE, HCAPLUS, BIOSIS, BIOTECHDS, EMBASE' ENTERED AT 17:51:52 ON 23 JUN 2006

L1 0 S DEAD GENE DISRUPTION AND CAROTENOID BIOSYNTHESIS
L2 0 S DEAD GENE AND CAROTENOID BIOSYNTHESIS
L3 0 S DEAD GENE AND ISOPRENOID BIOSYNTHESIS
L4 0 S DEAD GENE AND ISOPRENOID ENZYMATIC BIOSYNTHESIS
L5 0 S DEAD GENE AND ISOPRENOID
L6 41 S DEAD GENE
L7 19 DUP REM L6 (22 DUPLICATES REMOVED)
L8 3 S L7 AND (DISRUPT? OR DELETE)

=> d l7 1-19 ibib ab

L7 ANSWER 1 OF 19 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
ACCESSION NUMBER: 2006-03323 BIOTECHDS
TITLE: Increasing metabolic flux through the pentose phosphate pathway comprises culturing a microorganism comprising deregulated gene;
involving Corynebacterium glycerol-kinase, phosphoenolpyruvate-carboxykinese, glycogen-synthase, glucose-6-phosphate-insomerase, ATP-dependent RNA-helicase, succinylbenzoic acid-CoA-ligase, citrate lyase-beta chain, transcriptional regulator, pyruvate-dehydrogenase or succinyl-CoA-synthetase deregulated gene underexpression in Gram-pos. bacterium
AUTHOR: ZELDER O; KLOPPROGGE C; SCHROEDER H; HAEFNER S; KROEGER B; KIEFER P; HEINZLE E; WITTMANN C
PATENT ASSIGNEE: BASF AG
PATENT INFO: WO 2005121349 22 Dec 2005
APPLICATION INFO: WO 2004-IB4463 17 Dec 2004
PRIORITY INFO: WO 2003-IB646 18 Dec 2003; WO 2003-IB646 18 Dec 2003
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2006-056850 [06]
AB DERWENT ABSTRACT:

NOVELTY - Increasing metabolic flux through the pentose phosphate pathway in a microorganism comprises culturing a microorganism comprising a gene, which is deregulated under conditions such that metabolic flux through the pentose phosphate pathway is increased.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for a method for producing a fine chemical, and a recombinant microorganism, which has, or comprising a deregulated pentose phosphate biosynthesis pathway.

BIOTECHNOLOGY - Preferred Method: Fructose or sucrose is used as a carbon source. The gene is glycerol kinase, which is derived from Corynebacterium, and which is underexpressed. The gene encodes glycerol kinase, which has decreased activity. The microorganism is a Gram-positive microorganism. The microorganism belongs to the genus Corynebacterium, where the microorganism is Corynebacterium glutamicum. The microorganism is fermented to produce a fine chemical. The microorganism further comprises one or more additional deregulated gene. The one or more additional deregulated gene is an ask gene, a dapA gene, an asd gene, a dapB gene, a ddh gene, a lysA gene, a lysE gene, a pycA gene, a zwf gene, a pepCL gene, a gap gene, a zwa1 gene, a tkt gene, a tad gene, a mqo gene, a tpi gene, a pgk gene, or a sigC gene, where the one or more additional deregulated gene is overexpressed. The one or more additional deregulated gene encodes a protein selected from a feed-back resistant aspartokinase, a dihydrodipicolinate synthase, an aspartate semialdehyde dehydrogenase, a dihydrodipicolinate reductase, a diaminopimelate dehydrogenase, a diaminopimelate epimerase, a lysine exporter, a pyruvate carboxylase, a glucose-6-phosphate dehydrogenase, a phosphoenolpyruvate carboxylase, a glyceraldedyde-3-phosphate dehydrogenase, an RPF protein precursor, a transketolase, a transaldolase, a menaquinine oxidoreductase, a triosephosphate isomerase, a 3-phosphoglycerate kinase, or an RNA-polymerase sigma factor sigC, where the protein has increased activity. The one or more additional deregulated gene is a pepCK gene, a mal E gene, a glgA gene, a pgi gene, a **dead gene**, a menE gene, a citE gene, a mikE17 gene, a poxB gene, a zwa2 gene, or a sucC gene, where the one or more additional deregulated gene is attenuated, decreased or repressed. The one or more additional deregulated gene encodes a protein selected from a phosphoenolpyruvate carboxykinase, a malic enzyme, a glycogen synthase, a glucose-6-phosphate isomerase, an ATP dependent RNA helicase, an o-succinylbenzoic acid-CoA ligase, a citrate lyase beta chain, a transcriptional regulator, a pyruvate dehydrogenase, an RPF protein precursor, or a Succinyl-CoA-Synthetase, where the protein has decreased activity. Producing a fine chemical comprises culturing a microorganism

in which glycerol kinase is deregulated, and accumulating the fine chemical in the medium or in the cells of the microorganisms, thus producing a fine chemical. Alternatively, the method comprises culturing a microorganism in which at least one pentose phosphate biosynthetic pathway gene or enzyme is deregulated under conditions such that the fine chemical is produced. The method further comprises recovering the fine chemical. The fine chemical is lysine, where lysine is produced at a yield of at least 100 or 150 g/L. Glycerol kinase comprises the nucleotide sequence of 1650 bp (SEQ ID NO: 1). Glycerol kinase encodes a polypeptide comprising the sequence of 509 amino acids (SEQ ID NO: 2).

USE - The methods are useful for increasing metabolic flux through the pentose phosphate pathway in a microorganism and for producing a fine chemical.(90 pages)

L7 ANSWER 2 OF 19 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
ACCESSION NUMBER: 2005-20428 BIOTECHDS
TITLE: Increasing metabolic flux through pentose phosphate pathway in microorganism, useful for producing fine chemical e.g. lysine, by culturing microorganism having deregulated gene such that metabolic flux through pathway is increased; microorganism gene deregulation and pentose phosphate pathway-mediated increased metabolic flux for strain improvement, lysine preparation and fine chemical manufacture
AUTHOR: ZELDER O; KLOPPROGGE C; SCHROEDER H; HAEFNER S; KROEGER B; KIEFER P; HEINZLE E; WITTMANN C
PATENT ASSIGNEE: BASF AG
PATENT INFO: WO 2005059154 30 Jun 2005
APPLICATION INFO: WO 2004-IB4426 17 Dec 2004
PRIORITY INFO: WO 2003-IB6435 18 Dec 2003; WO 2003-IB6435 18 Dec 2003
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2005-479345 [48]
AB DERWENT ABSTRACT:

NOVELTY - Increasing (M1) metabolic flux through the pentose phosphate pathway in a microorganism, involves culturing a microorganism comprising a gene that is deregulated under conditions such that metabolic flux through the pentose phosphate pathway is increased.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for: (1) producing (M2) a fine chemical, involves culturing a microorganism in which lactate dehydrogenase is deregulated, and accumulating the fine chemical in the medium or in the cells of the microorganisms, or culturing microorganism in which a pentose phosphate biosynthetic pathway gene or enzyme is deregulated under conditions such that the fine chemical is produced; and (2) recombinant microorganism (I) which has a deregulated pentose phosphate biosynthetic pathway, or deregulated pentose phosphate biosynthesis gene.

BIOTECHNOLOGY - Preferred Method: In (M1), the fructose or sucrose, preferably fructose is used as a carbon source. The gene is lactate dehydrogenase derived from Corynebacterium. The lactate dehydrogenase gene is underexpressed. The gene encodes lactate dehydrogenase. The lactate dehydrogenase has decreased activity. The microorganism is a Gram-positive microorganism which microorganism belongs to the genus Corynebacterium, preferably C.glutamicum. The microorganism is fermented to produce a fine chemical and further comprises one or more additional deregulated gene chosen from an ask gene, dapA gene, an asd gene, dapB gene, ddh gene, lysA gene, lysE gene, pycA gene, zwf gene, pepCL gene, gap gene, zwa1 gene, tkt gene, tad gene, mqo gene, tpi gene, pgk gene, and sigC gene. The one or more additional deregulated gene is overexpressed. The additional deregulated gene encodes a protein chosen from feed-back resistant aspartokinase, dihydrodipicolinate synthase, aspartate semialdehyde dehydrogenase, dihydrodipicolinate reductase, diaminopimelate dehydrogenase, diaminopimelate epimerase, lysine exporter, pyruvate carboxylase, glucose-6-phosphate dehydrogenase, phosphoenolpyruvate carboxylase, glyceraldehydes-3-phosphate

dehydrogenase, an RPF protein precursor, transketolase, transaldolase, menaquinine oxidoreductase, triosephosphate isomerase, 3-phosphoglycerate kinase, and an RNA-polymerase sigma factor sigC. The protein has increased activity. The additional deregulated gene is chosen from pepCK gene, mal E gene, glgA gene, pgi gene, **dead gene**, menE gene, citE gene, mikE17 gene, poxB gene, zwa2 gene, and sucC gene. The additional deregulated gene is attenuated, decreased or repressed. The additional deregulated gene encodes a protein chosen from phosphoenolpyruvate carboxykinase, malic enzyme, glycogen synthase, glucose-6-phosphate isomerase, an ATP dependent RNA helicase, an o-succinylbenzoic acid-CoA ligase, citrate lyase beta chain, transcriptional regulator, pyruvate dehydrogenase, an RPF protein precursor, and a Succinyl-CoA-synthetase. The protein has decreased activity. In (M2), the biosynthetic gene and enzyme is lactate dehydrogenase whose expression or activity is increased. (M2) further involves recovering the fine chemical. The additional gene is deregulated. The fine chemical is lysine, which is produced at an yield of lysine is at least 100 g/L, or 150 g/L. The lactate dehydrogenase comprises a fully defined 1660 nucleotides (SEQ ID No. 1) sequence given in specification and encodes a polypeptide having a fully defined 420 amino acids (SEQ ID NO. 2) sequence given in the specification.

USE - (M1) is useful for increasing metabolic flux through pentose phosphate pathway in microorganism and thus for producing a fine chemical e.g. lysine (claimed).

ADVANTAGE - (M1) increases the production of the fine chemical e.g. lysine from a microorganism e.g. Corynebacterium by deregulating an enzyme encoding gene which is lactate dehydrogenase (claimed).(89 pages)

L7 ANSWER 3 OF 19 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 1

ACCESSION NUMBER: 2004:438652 BIOSIS
DOCUMENT NUMBER: PREV200400437476
TITLE: Genes of Helicobacter pylori regulated by attachment to AGS cells.
AUTHOR(S): Kim, Nayoung; Marcus, Elizabeth A.; Wen, Yi; Weeks, David L.; Scott, David R. [Reprint Author]; Jung, Hyun Chae; Song, In Sung; Sachs, George
CORPORATE SOURCE: VA Greater Los Angeles Hlth Care Syst, Bldg 113,Room 32A,11301 Wilshire Blvd, Los Angeles, CA, 90073, USA dscott@ucla.edu
SOURCE: Infection and Immunity, (April 2004) Vol. 72, No. 4, pp. 2358-2368. print.
ISSN: 0019-9567 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Nov 2004
Last Updated on STN: 17 Nov 2004

AB Reciprocal interactions between Helicobacter pylori and cells of the gastric epithelium to which it adheres may affect colonization. Changes in gene expression of H. pylori induced by adhesion to AGS gastric cancer cells by coculture were compared to changes in gene expression of H. pylori cultured without AGS cells by using cDNA filter macroarrays. Adhesion was quantitatively verified by confocal microscopy of green fluorescent protein-expressing bacteria. Four experiments showed that 22 and 21 H. pylori genes were consistently up- and down-regulated, respectively. The up-regulated genes included pathogenicity island, motility, outer membrane protein, and translational genes. The sigma28 factor antagonist flgM, flgG, the stress response gene,flaA, omp11, and the superoxide dismutase gene (sodB) were down-regulated. The up-regulation of cag3,flgB, tonB, rho, and deaD was confirmed by quantitative PCR, and the up-regulation of lpxD, omp6, secG, fabH, HP1285, HP0222, and HP0836 was confirmed by reverse transcription (RT)-PCR. The down-regulation of flaA, sodB, and HP0874 was confirmed by quantitative PCR, and the down-regulation of omp11 was confirmed by RT-PCR. The alteration of gene expression in H. pylori after adhesion to gastric cells

in vitro suggests that changes in motility, outer membrane composition, and stress responses, among other changes, may be involved in gastric colonization.

L7 ANSWER 4 OF 19 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:209240 HCAPLUS
DOCUMENT NUMBER: 141:406482
TITLE: Global expression analysis of the characterization of lysin production in Corynebacterium glutamicum
AUTHOR(S): Sindelar, Georg
CORPORATE SOURCE: Institut fuer Biotechnologie, Germany
SOURCE: Berichte des Forschungszentrums Juelich (2003), Juel-4092, 1-146
CODEN: FJBEE5; ISSN: 0944-2952
DOCUMENT TYPE: Report
LANGUAGE: German
AB New target genes and operons, resp. for the improvement of Lys prodn. by Corynebacterium glutamicum were identified applying genome-wide gene expression anal. by DNA chips. The gene expression patterns of a wild-type strain and of a potent prodn. strain MH20-22B obtained by mutagenesis were compared. The differences in the expression patterns were assigned to the deregulated aspartate kinase, to the Leu auxotrophy, and to further, unknown mutations. In C. glutamicum MH20-22B, 7 genes were up-regulated. Over-expression of the gene of a Me transferase of the uroporphyrin-II-C-Me transferase group, of a putative operon bearing the ammonium transporter Amt, of a putative Orn cyclodecarboxylase, and of a putative sarcosine oxidase caused an increase in Lys prodn. by 45%.
REFERENCE COUNT: 189 THERE ARE 189 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 5 OF 19 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 2002:256307 HCAPLUS
DOCUMENT NUMBER: 136:293612
TITLE: Sequence of **deaD gene** from corynebacteria and use thereof in synthesis of L-lysine
INVENTOR(S): Farwick, Mike; Huthmacher, Klaus; Brehme, Jennifer; Pfefferle, Walter
PATENT ASSIGNEE(S): Degussa A.-G., Germany
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002026787	A1	20020404	WO 2001-EP10772	20010918
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
DE 10047865	A1	20020418	DE 2000-10047865	20000927
AU 2001093821	A5	20020408	AU 2001-93821	20010918
EP 1320544	A1	20030625	EP 2001-974264	20010918
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2002115161	A1	20020822	US 2001-963790	20010927

PRIORITY APPLN. INFO.: DE 2000-10047865 A 20000927
WO 2001-EP10772 W 20010918

AB The **deaD gene** of Corynebacterium glutamicum ATCC13032 encoding a DNA/RNA helicase is cloned for use in increasing the efficiency of fermn. of L-lysine by coryneform bacteria. The expression vector contg. **deaD gene** is constructed. Methods and culture media for fermentative prepn. of L-lysine with recombinant bacterial strains transformed with these vectors are also provided. Disruption of the **deaD gene** by integration mutagenesis using deaD expression vector increased the yield of lysine in a Corynebacterium host from 13.57 g lysine/L at 7.6 OD660 to 16.31 g lysine/L at 12.2 OD660. The fermentatively prepd. L-lysine are useful in pharmaceutical industry and foodstuff industry and very particularly in animal nutrition.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 6 OF 19 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2000:347254 BIOSIS
DOCUMENT NUMBER: PREV200000347254
TITLE: Regulation of gene expression during aging.
AUTHOR(S): Helfand, Stephen L. [Reprint author]; Rogina, Blanka [Reprint author]
CORPORATE SOURCE: Department of BioStructure and Function, Universit of Connecticut Health Center, 263 Farmington Avenue, Farmington, CT, 06030, USA
SOURCE: Hekimi, Siegfried. Results Probl. Cell Differ., (2000) pp. 67-80. Results and Problems in Cell Differentiation; The molecular genetics of aging. print.
Publisher: Springer-Verlag, Heidelberger Pl 3, Berlin, Germany. Series: Results and Problems in Cell Differentiation.
CODEN: RCLDAT. ISSN: 0080-1844. ISBN: 3-540-66663-X (cloth).
DOCUMENT TYPE: Book
Book; (Book Chapter)
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Aug 2000
Last Updated on STN: 7 Jan 2002

L7 ANSWER 7 OF 19 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2000:408352 BIOSIS
DOCUMENT NUMBER: PREV200000408352
TITLE: Transcriptional and post-transcriptional control of polynucleotide phosphorylase during cold acclimation in Escherichia coli.
AUTHOR(S): Zangrossi, Sandro; Briani, Federica; Ghisotti, Daniela; Regonesi, Maria Elena; Tortora, Paolo; Deho, Gianni [Reprint author]
CORPORATE SOURCE: Dipartimento di Genetica e di Biologia dei Microrganismi, Universita degli Studi di Milano, Via Celoria 26, 20133, Milano, Italy
SOURCE: Molecular Microbiology, (June, 2000) Vol. 36, No. 6, pp. 1470-1480. print.
CODEN: MOMIEE. ISSN: 0950-382X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Sep 2000
Last Updated on STN: 8 Jan 2002

AB Polynucleotide phosphorylase (PNPase, polyribonucleotide nucleotidyltransferase, EC 2.7.7.8) is one of the cold shock-induced proteins in Escherichia coli and pnp, the gene encoding it, is essential for growth at low temperatures. We have analysed the expression of pnp upon cold shock and found a dramatic transient variation of pnp transcription profile: within the first hour after temperature downshift

the amount of pnp transcripts detectable by Northern blotting increased more than 10-fold and new mRNA species that cover pnp and the downstream region, including the cold shock gene deaD, appeared; 2 h after temperature downshift the transcription profile reverted to a preshift-like pattern in a PNPase-independent manner. The higher amount of pnp transcripts appeared to be mainly due to an increased stability of the RNAs. The abundance of pnp transcripts was not paralleled by comparable variation of the protein: PNPase steadily increased about twofold during the first 3 h at low temperature, as determined both by Western blotting and enzymatic activity assay, suggesting that PNPase, unlike other known cold shock proteins, is not efficiently translated in the acclimation phase. In experiments aimed at assessing the role of PNPase in autogenous control during cold shock, we detected a Rho-dependent termination site within pnp. In the cold acclimation phase, termination at this site depended upon the presence of PNPase, suggesting that during cold shock pnp is autogenously regulated at the level of transcription elongation.

L7 ANSWER 8 OF 19 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2000:260291 BIOSIS
DOCUMENT NUMBER: PREV200000260291
TITLE: Low temperature regulated DEAD-box RNA helicase from the Antarctic archaeon, Methanococcoides burtonii.
AUTHOR(S): Lim, Julianne; Thomas, Torsten; Cavicchioli, Ricardo [Reprint author]
CORPORATE SOURCE: School of Microbiology and Immunology, University of New South Wales, UNSW, Sydney, NSW, 2052, Australia
SOURCE: Journal of Molecular Biology, (March 31, 2000) Vol. 297, No. 3, pp. 553-567. print.
CODEN: JMOBAK. ISSN: 0022-2836.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Jun 2000
Last Updated on STN: 5 Jan 2002

AB DEAD-box RNA helicases, by unwinding duplex RNA in bacteria and eukaryotes, are involved in essential cellular processes, including translation initiation and ribosome biogenesis, and have recently been implicated in enabling bacteria to survive cold-shock and grow at low temperature. Despite these critical physiological roles, they have not been characterized in archaea. Due to their presumed importance in removing cold-stabilised secondary structures in mRNA, we have characterised a putative DEAD-box RNA helicase gene (deaD) from the Antarctic methanogen, Methanococcoides burtonii. The encoded protein, DeaD is predicted to contain a core element involved in ATP hydrolysis and RNA-binding, and an unusual C-terminal domain that contains seven perfect, tridecapeptide, direct repeats that may be involved in RNA binding. Alignment and phylogenetic analyses were performed on the core regions of the M. burtonii and other DEAD-box RNA helicases. These revealed a loose but consistent clustering of archaeal and bacterial sequences and enabled the generation of a prokaryotic-specific consensus sequence. The consensus highlights the importance of residues other than the eight motifs that are often associated with DEAD-box RNA helicases, as well as de-emphasising the importance of the "A" residue within the "DEAD" motif. Cells growing at 4degreeC contained abundant levels of deaD mRNA, however no mRNA was detected in cells growing at 23degreeC (the optimal temperature for growth). The transcription initiation site was mapped downstream from an archaeal box-A element (TATA box), which preceded a long (113 nucleotides) 5'-untranslated region (5'-UTR). Within the 5'-UTR was an 11 bp sequence that closely matches (nine out of 11) cold-box elements that are present in the 5'-UTRs of cold-shock induced genes from bacteria. To determine if the archaeal 5'-UTR performs an analagous function to the bacterial 5'-UTRs, the archaeal deaD 5'-UTR was transcribed in E. coli under the control of the cspA promoter and transcriptional terminator. It has previously been reported that overexpression of the cspA 5'-UTR leads to an extended cold-shock response

due to the 5'-UTR titrating cellular levels of a cold-shock repressor protein(s). In our hands, the cold-shock protein profiles resulting from overexpression of Escherichia coli cspA and M. burtonii deaD 5'-UTRs were similar, however they did not differ from those for the overexpression of a control plasmid lacking a 5'-UTR. In association with other recent data from E. coli, our results indicate that the role of the 5'-UTR in gene regulation is presently unclear. Irrespective of the mechanisms, it is striking that highly similar 5'-UTRs with cold-box elements are present in cold induced genes from E. coli, Anabaena and M. burtonii. This is the first study examining low temperature regulation in archaea and provides initial evidence that gene expression from a-cold adapted archaeon involves a bacterial-like transcriptional regulatory mechanism. In addition, it provides the foundation for further studies into the function and regulation of DEAD-box RNA helicases in archaea, and in particular, their roles in low temperature adaptation.

L7 ANSWER 9 OF 19 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1999:342538 BIOSIS
DOCUMENT NUMBER: PREV199900342538
TITLE: Identification and characterization of a new lipoprotein, NlpI, in Escherichia coli K-12.
AUTHOR(S): Ohara, Masaru; Wu, Henry C.; Sankaran, Krishnan; Rick, Paul D. [Reprint author]
CORPORATE SOURCE: Department of Microbiology and Immunology, Uniformed Services University of the Health Sciences, 4301 Jones Bridge Rd., Bethesda, MD, 20814-4799, USA
SOURCE: Journal of Bacteriology, (July, 1999) Vol. 181, No. 14, pp. 4318-4325. print.
CODEN: JOBAAY. ISSN: 0021-9193.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Aug 1999
Last Updated on STN: 24 Aug 1999
AB We report here the identification of a new lipoprotein, NlpI, in Escherichia coli K-12. The NlpI structural gene (nlpI) is located between the genes pnp (polynucleotide phosphorylase) and deaD (RNA helicase) at 71 min on the E. coli chromosome. The nlpI gene encodes a putative polypeptide of approximately 34 kDa, and multiple lines of evidence clearly demonstrate that NlpI is indeed a lipoprotein. An nlpI::cm mutation rendered growth of the cells osmotically sensitive, and incubation of the insertion mutant at an elevated temperature resulted in the formation of filaments. The altered phenotype of the mutant was a direct consequence of the mutation in nlpI, since it was complemented by the wild-type nlpI gene alone. Overexpression of the unaltered nlpI gene in wild-type cells resulted in the loss of the rod morphology and the formation of single prolate ellipsoids and pairs of prolate ellipsoids joined by partial constrictions. NlpI may be important for an as-yet-undefined step inthe overall process of cell division.

L7 ANSWER 10 OF 19 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1999:263505 BIOSIS
DOCUMENT NUMBER: PREV199900263505
TITLE: Identification of virulence genes of Helicobacter pylori by random insertion mutagenesis.
AUTHOR(S): Bijlsma, J. J. E.; Vandenbroucke-Grauls, C. M. J. E.; Phadnis, S. H.; Kusters, J. G. [Reprint author]
CORPORATE SOURCE: Department of Medical Microbiology, Faculty of Medicine, Vrije Universiteit Amsterdam, Van der Boechorststraat 7, 1081, BT Amsterdam, Netherlands
SOURCE: Infection and Immunity, (May, 1999) Vol. 67, No. 5, pp. 2433-2440. print.
CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
LANGUAGE: English

ENTRY DATE: Entered STN: 15 Jul 1999
Last Updated on STN: 15 Jul 1999

AB The complete genome of the gram-negative bacterial pathogen Helicobacter pylori, an important etiological agent of gastroduodenal disease in humans, has recently been published. This sequence revealed that the putative products of roughly one-third of the open reading frames (ORFs) have no significant homology to any known proteins. To be able to analyze the functions of all ORFs, we constructed an integration plasmid for H. pylori and used it to generate a random mutant library in this organism. This integration plasmid, designated pBCalpha3, integrated randomly into the chromosome of H. pylori. To test the capacity of this library to identify virulence genes, subsets of this library were screened for urease-negative mutants and for nonmotile mutants. Three urease-negative mutants in a subset of 1,251 mutants (0.25%) and 5 nonmotile mutants in a subset of 180 mutants (2.7%) were identified. Analysis of the disrupted ORFs in the urease-negative mutants revealed that two had disruptions of genes of the urease locus, ureB and ureI, and the third had a disruption of a unrelated gene; a homologue of deaD, which encodes an RNA helicase. Analysis of the disrupted ORFs in the nonmotile mutants revealed one ORF encoding a homologue of the paralyzed flagellar protein, previously shown to be involved in motility in Campylobacter jejuni. The other four ORFs have not been implicated in motility before. Based on these data, we concluded that we have generated a random insertion library in H. pylori that allows for the functional identification of genes in H. pylori.

L7 ANSWER 11 OF 19 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 1999232601 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10216955
TITLE: Molecular characterization of a prokaryotic translation factor homologous to the eukaryotic initiation factor eIF4A.
AUTHOR: Lu J; Aoki H; Ganoza M C
CORPORATE SOURCE: Banting and Best Department of Medical Research, University of Toronto, Ont., Canada.
SOURCE: The international journal of biochemistry & cell biology, (1999 Jan) Vol. 31, No. 1, pp. 215-29.
Journal code: 9508482. ISSN: 1357-2725.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199905
ENTRY DATE: Entered STN: 1 Jun 1999
Last Updated on STN: 1 Jun 1999
Entered Medline: 17 May 1999

AB Initiation of translation involves a complex series of reactions that result in the formation of an initiation complex at the proper start site of the mRNA. These reactions, particularly those that involve the binding of the mRNA to the small subunit of the ribosome, are not fully understood. Here we show that one of the factors (W2) required to reconstitute translation in E. coli is encoded by the **deaD gene** which harbors 87% amino acid sequence similarly to the eukaryotic (eIF4A). Antibodies against the eukaryotic eIF4A cross-react with the E. coli protein. We describe the overexpression of the W2 protein from recombinant clones and its purification in one step by the use of a His tag at the N-terminus of its sequence. We report a rapid assay for the W2 protein that scores for initiation and elongation programmed by a native mRNA template. The W2 protein promotes initiation programmed by the mRNA that harbors secondary structures. The W2 protein is not required in standard initiation assays programmed by synthetic mRNAs of defined sequence that lack this feature. We conclude that W2 is an important factor for initiation in eukaryotic and prokaryotic cells.

L7 ANSWER 12 OF 19 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 97322367 MEDLINE

DOCUMENT NUMBER: PubMed ID: 9177212
TITLE: Drosophila drop-dead mutations accelerate the time course of age-related markers.
AUTHOR: Rogina B; Benzer S; Helfand S L
CORPORATE SOURCE: Department of BioStructure and Function, School of Dental Medicine, University of Connecticut Health Center, Farmington, CT 06030, USA.
CONTRACT NUMBER: AG 12289 (NIA)
EY 09278 (NEI)
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1997 Jun 10) Vol. 94, No. 12, pp. 6303-6.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 21 Jul 1997
Last Updated on STN: 21 Jul 1997
Entered Medline: 10 Jul 1997

AB Mutations of the drop-**dead gene** in Drosophila melanogaster lead to striking early death of the adult animal. At different times, after emergence from the pupa, individual flies begin to stagger and, shortly thereafter, die. Anatomical examination reveals gross neuropathological lesions in the brain. The life span of flies mutant for the drop-**dead gene** is four to five times shorter than for normal adults. That raises the question whether loss of the normal gene product might set into motion a series of events typical of the normal aging process. We used molecular markers, the expression patterns of which, in normal flies, change with age in a manner that correlates with life span. In the drop-dead mutant, there is an acceleration in the temporal pattern of expression of these age-related markers.

L7 ANSWER 13 OF 19 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1997:64814 BIOSIS
DOCUMENT NUMBER: PREV199799364017
TITLE: Sequencing of a 65 kb region of the Bacillus subtilis genoma containing the lic and cel loci, and creation of a 177 kb contig covering the gnt-sacXY region.
AUTHOR(S): Yoshida, Ken-Ichi; Shindo, Katsuhiro; Sano, Hidetoshi; Seki, Shin; Fujimura, Miyuki; Yanai, Nobuo; Miwa, Yasuhiko; Fujita, Yasutaro [Reprint author]
CORPORATE SOURCE: Dep. Biotechnol., Fac. Eng., Fukuyama University, Higashimura-cho, Fukuyama-shi, Hiroshima 729-02, Japan
SOURCE: Microbiology (Reading), (1996) Vol. 142, No. 11, pp. 3113-3123.
ISSN: 1350-0872.
DOCUMENT TYPE: Article
LANGUAGE: English
OTHER SOURCE: DDBJ-D83026; EMBL-D83026
ENTRY DATE: Entered STN: 11 Feb 1997
Last Updated on STN: 25 Mar 1997

AB Within the framework of an international project for the sequencing of the entire Bacillus subtilis genome, this paper communicates the sequencing of a chromosome region containing the lic and cel loci (65 kb), which creates a 177 kb contig covering the region from gnt to sacXY. This 65 kb region contains 64 ORFs (62 complete and two partial genes). The 14th, 15th and 17th genes correspond to licT, licS and katE, encoding the antiterminator for licS transcription, beta-glucanase (lichenase) and catalase 2, respectively. The 11th, 30th, 36th, 39th, 41st, 45th-48th, 51st and 58th genes are designated deaD, pepT, galE, aldY, msmX, cydABCD, sigY and katX because their products probably encode ATP-dependent RNA helicase,

tripeptidase, UDP-glucose 4-epimerase, aldehyde dehydrogenase, multiple sugar-binding transport ATP-binding protein, the respective components of cytochrome d ubiquinol oxidase and ATP-binding cassette transporter, sigma-factor of RNA polymerase and catalase, respectively. The 60th-64th genes are celRABCD, which are probably involved in cellobiose utilization. Gene organization and gene features in the gnt-sacXY region are discussed.

L7 ANSWER 14 OF 19 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 96133880 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8552679
TITLE: Cold shock induces a major ribosomal-associated protein that unwinds double-stranded RNA in Escherichia coli.
AUTHOR: Jones P G; Mitta M; Kim Y; Jiang W; Inouye M
CORPORATE SOURCE: Department of Biochemistry, University of Medicine and Dentistry of New Jersey, Robert Wood Johnson Medical School, Piscataway 08854, USA.
CONTRACT NUMBER: GM19043 (NIGMS)
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1996 Jan 9) Vol. 93, No. 1, pp. 76-80.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199602
ENTRY DATE: Entered STN: 6 Mar 1996
Last Updated on STN: 6 Mar 1996
Entered Medline: 22 Feb 1996
AB A 70-kDa protein was specifically induced in Escherichia coli when the culture temperature was shifted from 37 to 15 degrees C. The protein was identified to be the product of the **deaD gene** (reassigned csdA) encoding a DEAD-box protein. Furthermore, after the shift from 37 to 15 degrees C, CsdA was exclusively localized in the ribosomal fraction and became a major ribosomal-associated protein in cells grown at 15 degrees C. The csdA deletion significantly impaired cell growth and the synthesis of a number of proteins, specifically the derepression of heat-shock proteins, at low temperature. Purified CsdA was found to unwind double-stranded RNA in the absence of ATP. Therefore, the requirement for CsdA in derepression of heat-shock protein synthesis is a cold shock-induced function possibly mediated by destabilization of secondary structures previously identified in the rpoH mRNA.

L7 ANSWER 15 OF 19 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1995:292497 BIOSIS
DOCUMENT NUMBER: PREV199598306797
TITLE: A deaD-box protein: A cold-shock ribosomal protein required for optimal gene expression and growth at low temperature in E. coli.
AUTHOR(S): Jones, Pamela G. [Reprint author]; Kim, Young-Ho; Jiang, Weining; Inouye, Masayori
CORPORATE SOURCE: Robert Wood Johnson Med. Sch., Piscataway, NJ 08854, USA
SOURCE: Abstracts of the General Meeting of the American Society for Microbiology, (1995) Vol. 95, No. 0, pp. 562.
Meeting Info.: 95th General Meeting of the American Society for Microbiology. Washington, D.C., USA. May 21-25, 1995.
ISSN: 1060-2011.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Jul 1995
Last Updated on STN: 5 Jul 1995

L7 ANSWER 16 OF 19 MEDLINE on STN DUPLICATE 6

ACCESSION NUMBER: 94334279 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8056751
TITLE: Nucleotide sequence and expression in Escherichia coli of the Klebsiella pneumoniae **deaD gene**.
AUTHOR: Peng H L; Hsieh M J; Zao C L; Chang H Y
CORPORATE SOURCE: Department of Microbiology and Immunology, Chang-Gung Medical College, Kwei-San, Taiwan.
SOURCE: Journal of biochemistry, (1994 Mar) Vol. 115, No. 3, pp. 409-14.
Journal code: 0376600. ISSN: 0021-924X.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L08387; GENBANK-U03750
ENTRY MONTH: 199409
ENTRY DATE: Entered STN: 20 Sep 1994
Last Updated on STN: 20 Sep 1994
Entered Medline: 14 Sep 1994

AB The **deaD gene** of Klebsiella pneumoniae was isolated and its nucleotide sequence determined. The K. pneumoniae gene is highly homologous with the Escherichia coli analog throughout most of the coding region. The deduced primary sequence of the K. pneumoniae **deaD gene** product is 659 amino acids in length, in contrast with the 571 amino acids of the E. coli deaD product published previously. Sequence comparison revealed several differences near the 3' end of the deaD genes which result in the frame-shift effect. The 3' end sequence of the E. coli **deaD gene** was therefore analyzed to verify the discrepancy. Our result indicates that the E. coli **deaD gene** encodes a product of comparable size to the K. pneumoniae DeaD protein, and the carboxyl terminal sequences of the two proteins are highly homologous. In vivo expression of the K. pneumoniae **deaD gene** in E. coli yielded a 65-kDa protein. Primer extension analysis of the mRNA from K. pneumoniae identified a major transcription start site at an A residue 44 nt upstream of the first in-frame ATG codon.

L7 ANSWER 17 OF 19 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 94247361 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8190075
TITLE: Multicopy suppressors, mssA and mssB, of an smbA mutation of Escherichia coli.
AUTHOR: Yamanaka K; Ogura T; Koonin E V; Niki H; Hiraga S
CORPORATE SOURCE: Department of Molecular Cell Biology, Kumamoto University School of Medicine, Japan.
SOURCE: Molecular & general genetics : MGG, (1994 Apr) Vol. 243, No. 1, pp. 9-16.
Journal code: 0125036. ISSN: 0026-8925.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: SWISSPROT-P05082; SWISSPROT-P07170; SWISSPROT-P10251; SWISSPROT-P10772; SWISSPROT-P12115; SWISSPROT-P15700; SWISSPROT-P16304; SWISSPROT-P20425; SWISSPROT-P25824; SWISSPROT-P26364; SWISSPROT-P27134; SWISSPROT-P27144
ENTRY MONTH: 199406
ENTRY DATE: Entered STN: 29 Jun 1994
Last Updated on STN: 6 Apr 2003
Entered Medline: 17 Jun 1994

AB We have isolated and characterized two multicopy suppressors, mssA and mssB, which suppress the cold-sensitive growth phenotype of the smbA2 mutant of Escherichia coli. The mssA gene is located immediately upstream of the rpsA gene (20.5 min). MssA protein was found to be related to nucleoside monophosphate kinases. The mssB gene was found to be identical to the **deaD gene** (69 min), which encodes a putative

RNA helicase. The SmbA protein belongs to the aspartokinase family and probably represents a new, fourth aspartokinase species in E. coli. Expression of the smbA gene is essential for cell growth. The smbA2 mutant shows a pleiotropic phenotype characterized by cold-sensitive growth, hypersensitivity to the detergent sodium dodecyl sulfate, and formation of a translucent segment at midcell or at a pole of the cell when grown at 22 degrees C. In addition, some cellular proteins were either increased or decreased in amount in the smbA2 mutant. SmbA may be a regulatory factor in the expression of a battery of genes. MssA and MssB might also relate to the expression of some of these genes. Multiple copies mssA and mssB suppressed the various phenotypic features of the smbA2 mutant to various extents, suppressing the cold-sensitive growth completely.

L7 ANSWER 18 OF 19 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 93264080 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8494644
TITLE: Defective glia in the Drosophila brain degeneration mutant drop-dead.
AUTHOR: Buchanan R L; Benzer S
CORPORATE SOURCE: California Institute of Technology, Pasadena, California 91125.
CONTRACT NUMBER: 5F32NS0881-02 (NINDS)
EY09278 (NEI)
GM 40499 (NIGMS)
SOURCE: Neuron, (1993 May) Vol. 10, No. 5, pp. 839-50.
Journal code: 8809320. ISSN: 0896-6273.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199306
ENTRY DATE: Entered STN: 2 Jul 1993
Last Updated on STN: 29 Jan 1996
Entered Medline: 18 Jun 1993
AB To understand better the cellular basis of late-onset neuronal degeneration, we have examined the brain of the drop-dead mutant of Drosophila. This mutant carries an X-chromosomal recessive mutation that causes severe behavioral defects and brain degeneration, manifested a few days after emergence of the adult. Analysis of genetically mosaic flies has indicated that the focus of the drop-dead mutant phenotype is in the brain and that the gene product is non-cell autonomous. We examined the adult drop-dead mutant brain prior to onset of symptoms and found that many glial cells have stunted processes, whereas neuronal morphology is essentially normal. Adult mutant glial cells resemble immature glia found at an earlier stage of normal brain development. These observations suggest that defective glia in the drop-dead brain may disrupt adult nervous system function, contributing to progressive brain degeneration and death. The normal drop-**dead gene** product may prevent brain degeneration by providing a necessary glial function.

L7 ANSWER 19 OF 19 MEDLINE on STN DUPLICATE 9
ACCESSION NUMBER: 94032476 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7692973
TITLE: Dbp45A encodes a Drosophila DEAD box protein with similarity to a putative yeast helicase involved in ribosome assembly.
AUTHOR: Lavoie C A; Harvey M; Lasko P F
CORPORATE SOURCE: Department of Biology, McGill University, Montreal, Quebec, Canada.
SOURCE: Biochimica et biophysica acta, (1993 Oct 19) Vol. 1216, No. 1, pp. 140-4.
Journal code: 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-D12632; GENBANK-D12633; GENBANK-L08438; GENBANK-L08439; GENBANK-L08440; GENBANK-L08441; GENBANK-L08442; GENBANK-L08443; GENBANK-L13612; GENBANK-X61053
ENTRY MONTH: 199312
ENTRY DATE: Entered STN: 17 Jan 1994
Last Updated on STN: 29 Jan 1996
Entered Medline: 2 Dec 1993

AB Proteins of the DEAD family of putative ATP-dependent RNA helicases have been implicated in translation initiation, ribosome assembly, and RNA processing in a variety of organisms from Escherichia coli to man. Among these proteins are eIF-4A, an essential component of the cap-binding complex, numerous yeast proteins required for pre-mRNA splicing, and proteins from yeast and E. coli necessary for ribosome assembly. We report the isolation of a new **DEAD gene** from Drosophila, Dbp45A, which is most abundantly expressed in 6-12 h embryos and adults. The predicted amino acid sequence of the Dbp45A product contains all eight highly conserved DEAD family motifs, and most closely resembles the Saccharomyces cerevisiae DRS1p among known DEAD box proteins. DRS1p has been implicated in ribosomal RNA processing.

=> d his

(FILE 'HOME' ENTERED AT 17:51:22 ON 23 JUN 2006)

FILE 'MEDLINE, HCAPLUS, BIOSIS, BIOTECHDS, EMBASE' ENTERED AT 17:51:52 ON 23 JUN 2006

L1 0 S DEAD GENE DISRUPTION AND CAROTENOID BIOSYNTHESIS
L2 0 S DEAD GENE AND CAROTENOID BIOSYNTHESIS
L3 0 S DEAD GENE AND ISOPRENOID BIOSYNTHESIS
L4 0 S DEAD GENE AND ISOPRENOID ENZYMATIC BIOSYNTHESIS
L5 0 S DEAD GENE AND ISOPRENOID
L6 41 S DEAD GENE
L7 19 DUP REM L6 (22 DUPLICATES REMOVED)
L8 3 S L7 AND (DISRUPT? OR DELETE)

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☐ 1. Document ID: US 20040265861 A1

L2: Entry 1 of 17 File: PGPB Dec 30, 2004

PGPUB-DOCUMENT-NUMBER: 20040265861
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20040265861 A1

TITLE: Materials and methods for identifying genes and/or agents that alter replicative lifespan

PUBLICATION-DATE: December 30, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Goldfarb, David S.	Victor	NY	US

US-CL-CURRENT: 435/6; 435/254.2, 435/483

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 2. Document ID: US 20040191863 A1

L2: Entry 2 of 17 File: PGPB Sep 30, 2004

PGPUB-DOCUMENT-NUMBER: 20040191863
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20040191863 A1

TITLE: Mutations affecting plasmid copy number

PUBLICATION-DATE: September 30, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Cheng, Qiong	Wilmington	DE	US
Rouviere, Pierre E.	Wilmington	DE	US
Tao, Luan	Claymont	DE	US
Suh, Wonchul	Hockessin	DE	US

US-CL-CURRENT: 435/69.1; 435/252.3, 435/252.33, 435/471, 435/488

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 3. Document ID: US 20040146966 A1

L2: Entry 3 of 17 File: PGPB Jul 29, 2004

PGPUB-DOCUMENT-NUMBER: 20040146966
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20040146966 A1

TITLE: Mutations affecting carotenoid production

PUBLICATION-DATE: July 29, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Cheng, Qiong	Wilmington	DE	US
Rouviere, Pierre E.	Wilmington	DE	US
Tao, Luan	Claymont	DE	US

US-CL-CURRENT: 435/67; 435/252.3, 435/254.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWMC | Draw Desc | Image

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☐ 4. Document ID: US 20040033586 A1

L2: Entry 4 of 17 File: PGPB Feb 19, 2004

PGPUB-DOCUMENT-NUMBER: 20040033586
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20040033586 A1

TITLE: Attenuated gram negative bacteria

PUBLICATION-DATE: February 19, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Crooke, Helen Rachel	Winnersh Triangle		GB
Shea, Jacqueline Elizabeth	Winnersh Triangle		GB
Feldman, Robert Graham	Winnersh Triangle		GB
Goutebroze, Sylvain Gabriel	Lyon		FR
Le Gros, Francois-Xavier	Saint Genis Laval		FR

US-CL-CURRENT: 435/252.3

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWMC | Draw Desc | Image

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☐ 5. Document ID: US 20020115161 A1

L2: Entry 5 of 17 File: PGPB Aug 22, 2002

PGPUB-DOCUMENT-NUMBER: 20020115161
PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20020115161 A1

TITLE: Nucleotide sequences which code for the deaD gene

PUBLICATION-DATE: August 22, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Farwick, Mike	Bielefeld		DE
Huthmacher, Klaus	Gelnhausen		DE
Brehme, Jennifer	Bielefeld		DE
Pfefferle, Walter	Halle		DE

US-CL-CURRENT: 435/115; 435/219, 435/252.3, 435/320.1, 435/69.1, 536/23.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Drawi Desc | Image

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☐ 6. Document ID: US 6703484 B2

L2: Entry 6 of 17 File: USPT Mar 9, 2004

US-PAT-NO: 6703484
DOCUMENT-IDENTIFIER: US 6703484 B2

TITLE: Methods for production of proteins

DATE-ISSUED: March 9, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Chatterjee; Deb	North Potomac	MD		
Longo; Mary	Germantown	MD		
Flynn; Elizabeth	Columbia	MD		
Oberfelder; Robert	Woodland	TX		

US-CL-CURRENT: 530/350; 435/68.1, 435/69.1, 435/69.7

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Drawi Desc | Image

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☐ 7. Document ID: US 6551795 B1

L2: Entry 7 of 17 File: USPT Apr 22, 2003

US-PAT-NO: 6551795
DOCUMENT-IDENTIFIER: US 6551795 B1

TITLE: Nucleic acid and amino acid sequences relating to pseudomonas aeruginosa for diagnostics and therapeutics

DATE-ISSUED: April 22, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Rubenfield; Marc J.	Framingham	MA		
Nolling; Jork	Ouincy	MA		
Deloughery; Craig	Medford	MA		
Bush; David	Somerville	MA		

US-CL-CURRENT: 435/69.1; 435/253.3, 435/320.1, 435/325, 435/6, 536/23.1, 536/23.7

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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☐ 8. Document ID: US 6020149 A

L2: Entry 8 of 17 File: USPT Feb 1, 2000

US-PAT-NO: 6020149
DOCUMENT-IDENTIFIER: US 6020149 A

TITLE: Methods of screening for anti-microbial agents and for inhibiting microbial growth

DATE-ISSUED: February 1, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Fuller-Pace; Frances Victoria	Fife			GB
Lane; David Philip	Fife			GB

US-CL-CURRENT: 435/32; 435/21

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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☐ 9. Document ID: JP 08129586 A

L2: Entry 9 of 17 File: JPAB May 21, 1996

PUB-NO: JP408129586A
DOCUMENT-IDENTIFIER: JP 08129586 A
TITLE: SCHEDULE GENERATING METHOD

PUBN-DATE: May 21, 1996

INVENTOR-INFORMATION:

NAME	COUNTRY
MORIKAWA, MASASHI	

INT-CL (IPC): G06 F 17/60; B23 Q 41/08

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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☐ 10. Document ID: WO 200226787 A1, DE 10047865 A1, AU 200193821 A, US 20020115161 A1, EP

. 1320544 A1

L2: Entry 10 of 17 File: DWPI Apr 4, 2002

DERWENT-ACC-NO: 2002-394238
DERWENT-WEEK: 200550

TITLE: New deaD gene encoding polypeptide having activity of DNA/RNA helicase, useful in bacteria for the fermentative preparation of L-amino acids, particularly L-lysine, from glucose, molasses, starch, cellulose or ethanol

INVENTOR: BREHME, J; FARWICK, M ; HUTHMACHER, K ; PFEFFERLE, W

PRIORITY-DATA: 2000DE-1047865 (September 27, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 200226787 A1	April 4, 2002	E	052	C07K014/34
DE 10047865 A1	April 18, 2002		000	C12N001/21
AU 200193821 A	April 8, 2002		000	C07K014/34
US 20020115161 A1	August 22, 2002		000	C12P013/08
EP 1320544 A1	June 25, 2003	E	000	C07K014/34

INT-CL (IPC): C07 H 21/00; C07 H 21/04; C07 K 14/34; C12 N 1/21; C12 N 9/00; C12 N 9/50; C12 N 15/10; C12 N 15/52; C12 N 15/63; C12 N 15/74; C12 P 13/04; C12 P 13/08; C12 P 13:08; C12 P 21/02; C12 Q 1/68; C12 R 1:15; C12 R 1:15; C12 P 13/08

Terms	Documents
deaD gene	17

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L2: Entry 5 of 17 File: PGPB Aug 22, 2002

PGPUB-DOCUMENT-NUMBER: 20020115161
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020115161 A1

TITLE: Nucleotide sequences which code for the deaD gene

PUBLICATION-DATE: August 22, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Farwick, Mike	Bielefeld		DE
Huthmacher, Klaus	Gelnhausen		DE
Brehme, Jennifer	Bielefeld		DE
Pfefferle, Walter	Halle		DE

APPL-NO: 09/963790 [PALM]
DATE FILED: September 27, 2001

FOREIGN-APPL-PRIORITY-DATA:

COUNTRY	APPL-NO	DOC-ID	APPL-DATE
DE	100 47 865.4	2000DE-100 47 865.4	September 27, 2000

INT-CL-PUBLISHED: [07] C12 P 13/08, C07 H 21/04, C12 N 9/50, C12 P 21/02, C12 N 1/21, C12 N 15/74

US-CL-PUBLISHED: 435/115; 435/219, 435/69.1, 435/252.3, 435/320.1, 536/23.2
US-CL-CURRENT: 435/115; 435/219, 435/252.3, 435/320.1, 435/69.1, 536/23.2

REPRESENTATIVE-FIGURES: 1

ABSTRACT:

The invention relates to an isolated polynucleotide having a polynucleotide sequence which codes for the deaD gene, and a host-vector system having a coryneform host bacterium in which the deaD gene is present in attenuated form and a vector which carries at least the deaD gene according to SEQ ID No 1, and the use of polynucleotides which comprise the sequences according to the invention as hybridization probes.

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[0067] A common method of mutating genes of C. glutamicum is the method of "gene disruption" and "gene replacement" described by Schwarzer and Puhler (Bio/Technology 9, 84-87 (1991)) I.B.R.

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L8: Entry 2 of 2 File: PGPB Jul 29, 2004

PGPUB-DOCUMENT-NUMBER: 20040146966
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20040146966 A1

TITLE: Mutations affecting carotenoid production

PUBLICATION-DATE: July 29, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Cheng, Qiong	Wilmington	DE	US
Rouviere, Pierre E.	Wilmington	DE	US
Tao, Luan	Claymont	DE	US

APPL-NO: 10/735008 [PALM]
DATE FILED: December 12, 2003

RELATED-US-APPL-DATA:
Application is a non-provisional-of-provisional application 60/435612, filed December 19, 2002,

INT-CL-PUBLISHED: [07] C12 P 23/00, C12 N 1/21, C12 N 1/16, C12 N 1/18

US-CL-PUBLISHED: 435/067; 435/252.3, 435/254.2
US-CL-CURRENT: 435/67; 435/252.3, 435/254.2

REPRESENTATIVE-FIGURES: NONE

ABSTRACT:

Mutations in genes having no direct relationship to the carotenoid biosynthetic pathway have been found to increase carbon flux through that pathway. Complete disruption in the deaD, mreC, and yfhE genes were effective. Additionally where genes of the lower carotenoid pathway reside on a plasmid having either a p15A or pMB1 replicon, mutations in the thrS, rspA, rpoC, yjeR, and rhoL were found effective.

[0001] This application claims the benefit of U.S. Provisional Application No. 60/435,612 filed Dec. 19, 2002.

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WEST Search History

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DATE: Friday, June 23, 2006

Hide?	Set Name	Query	Hit Count
	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L8	deaD gene and carotenoid biosynthesis	2
☐	L7	deaD gene and disruption	7
☐	L6	deaD gene with disruption	0
☐	L5	deaD gene same disruption	0
	DB=EPAB; PLUR=YES; OP=ADJ		
☐	L4	WO-9325685-A1.did.	1
	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L3	deaD gene disruption	0
☐	L2	deaD gene	17
☐	L1	deaD gene deletion	0

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